We Claim:

1. A chimeric protein comprising a first and a second polypeptide wherein the first polypeptide is a Factor VII or Factor VIIa polypeptide and the second polypeptide is an Fc region of a human immunoglobulin IgG1, wherein the Factor VII or Factor VIIa polypeptide contains at least one mutant residue which prevents proteolytic cleavage between residues 38 and 39 or between residues 152 and 153.
2. The chimeric protein of claim 1 wherein the mutant residue is selected from the group consisting of amino acid residue 38 and amino acid residue 152, wherein the amino acid residue at position 38 is not a lysine and the amino acid residue at position 152 is not an arginine.
3. The chimeric protein of claim 1or 2 wherein the mutant residue is an alanine.
4. The chimeric protein of claim 2 wherein the mutant residue is a glutamine at residue 152.
5. The chimeric protein of claim 2 wherein the mutant residue is a glutamate at residue 152.
6. The chimeric protein of claim 1 wherein the Factor VII or Factor VIIa polypeptide contains an active site mutation which when present in Factor VIIa reduces blood coagulation activity relative to wild-type Factor VIIa.
7. The chimeric protein of claim 6 wherein the active site mutation is selected from the group consisting of: a non-lysine at residue 341, a non-serine residue at residue 344 and combinations thereof.
8. The chimeric protein of claim 6 wherein the active site mutation is an alanine substitution.
9. The chimeric protein of claim 1 wherein the second polypeptide comprises at least one mutation in a residue selected from the group consisting of K326 and E333 as denominated in an intact immunoglobulin, wherein the mutation increases the binding of the second polypeptide to complement constituent C1q.
10. The chimeric protein of claim 9 wherein the mutation in the second polypeptide is a tryptophan residue at K326.

11. The chimeric protein of claim 9 wherein the mutation in the second polypeptide is a serine residue at E333.
12. The chimeric protein of claim 9 wherein the second polypeptide comprises two of said mutations.
13. The chimeric protein of claim 1 which is in the form of a dimer.
14. A method of treating a patient having disease associated with neovascularization comprising:

    administering to the patient an effective amount of a chimeric protein comprising a first and a second polypeptide wherein the first polypeptide is a Factor VII or Factor VIIa polypeptide and the second polypeptide is an Fc region of a human immunoglobulin IgG1, wherein the Factor VII or Factor VIIa polypeptide contains at least one mutant residue which prevents proteolytic cleavage between residues 38 and 39 or between residues 152 and 153, whereby symptoms of the disease are ameliorated.
15. The method of claim 14 wherein the disease is cancer.
16. The method of claim 14 wherein the disease is wet macular degeneration.
17. The method of claim 14 wherein the mutant residue is selected from the group consisting of amino acid residue 38 and amino acid residue 152, wherein the amino acid residue at position 38 is not a lysine and the amino acid residue at position 152 is not an arginine.
18. The method of claim 17 wherein the mutant residue is an alanine.
19. The method of claim 17 wherein the mutant residue is a glutamine at residue 152.
20. The method of claim 17 wherein the mutant residue is a glutamate at residue 152.
21. The method of claim 14 wherein the Factor VII or Factor VIIa polypeptide contains an active site mutation which when present in Factor VIIa reduces blood coagulation activity relative to wild-type Factor VIIa.
22. The method of claim 21 wherein the active site mutation is selected from the group consisting of: a non-lysine at residue 341, a non-serine residue at residue 344 and combinations thereof.
23. The method of claim 21 wherein the active site mutation is an alanine substitution.
24. The method of claim 14 wherein the second polypeptide comprises at least one mutation in a residue selected from the group consisting of K326 and E333 as

denominated in an intact immunoglobulin, wherein the mutation increases the binding of the second polypeptide to complement constituent C1q.

25. The method of claim 24 wherein the mutation in the second polypeptide is a tryptophan residue at K326.
26. The method of claim 24 wherein the mutation in the second polypeptide is a serine residue at E333.
27. The method of claim 24 wherein the second polypeptide comprises two of said mutations.
28. An expression vector that encodes a secreted form of a chimeric protein comprising a first and a second polypeptide wherein the first polypeptide is a Factor VII or Factor VIIa polypeptide and the second polypeptide is an Fc region of a human immunoglobulin IgG1, wherein the Factor VII or Factor VIIa polypeptide contains at least one mutant residue that prevents proteolytic cleavage between residues 38 and 39 or between residues 152 and 153.
29. The expression vector of claim 28 which is a replication-deficient adenoviral vector or adeno-associated vector.
30. The expression vector of claim 28 wherein the mutant residue is selected from the group consisting of amino acid residue 38 and amino acid residue 152, wherein the amino acid residue at position 38 is not a lysine and the amino acid residue at position 152 is not an arginine.
31. The expression vector of claim 30 wherein the mutant residue is an alanine.
32. The expression vector of claim 30 wherein the mutant residue is a glutamine at residue 152.
33. The method of claim 30 wherein the mutant residue is a glutamate at residue 152.
34. The expression vector of claim 28 wherein the Factor VII or Factor VIIa polypeptide contains an active site mutation which when present in Factor VIIa reduces blood coagulation activity relative to wild-type Factor VIIa.
35. The method of claim 34 wherein the active site mutation is selected from the group consisting of: a non-lysine at residue 341, a non-serine residue at residue 344 and combinations thereof.
36. The expression vector of claim 34 wherein the active site mutation is an alanine substitution.

37. The expression vector of claim 28 wherein the second polypeptide comprises at least one mutation in a residue selected from the group consisting of K326 and E333 as denominated in an intact immunoglobulin, wherein the mutation increases the binding of the second polypeptide to complement constituent C1q.
38. The expression vector of claim 37 wherein the mutation in the second polypeptide is a tryptophan residue at K326.
39. The expression vector of claim 37 wherein the mutation in the second polypeptide is a serine residue at E333.
40. The expression vector of claim 37 wherein the second polypeptide comprises two of said mutations.
41. A method of treating a patient having disease associated with neovascularization comprising:

    administering to the patient an effective amount of an expression vector encoding a secreted form of a chimeric protein comprising a first and a second polypeptide wherein the first polypeptide is a Factor VII or Factor VIIa polypeptide and the second polypeptide is an Fc region of a human immunoglobulin IgG1, wherein the Factor VII or Factor VIIa polypeptide contains at least one mutant residue which prevents proteolytic cleavage between residues 38 and 39 or between residues 152 and 153, whereby symptoms of the disease are ameliorated.
42. The method of claim 41 wherein the disease is cancer.
43. The method of claim 41 wherein the disease is wet macular degeneration.
44. The method of claim 41 wherein the mutant residue is selected from the group consisting of amino acid residue 38 and amino acid residue 152, wherein the amino acid residue at position 38 is not a lysine and the amino acid residue at position 152 is not an arginine.
45. The method of claim 44 wherein the mutant residue is an alanine.
46. The method of claim 44 wherein the mutant residue is a glutamine at residue 152.
47. The method of claim 44 wherein the mutant residue is a glutamate at residue 152.
48. The method of claim 41 wherein the Factor VII or Factor VIIa polypeptide contains an active site mutation which when present in Factor VIIa reduces blood coagulation activity relative to wild-type Factor VIIa.

49. The method of claim 48 wherein the active site mutation is selected from the group consisting of: a non-lysine at residue 341, a non-serine residue at residue 344 and combinations thereof.
50. The method of claim 48 wherein the active site mutation is an alanine substitution.
51. The method of claim 41 wherein the second polypeptide comprises at least one mutation in a residue selected from the group consisting of K326 and E333 as denominated in an intact immunoglobulin, wherein the mutation increases the binding of the second polypeptide to complement constituent C1q.
52. The method of claim 51 wherein the mutation in the second polypeptide is a tryptophan residue at K326.
53. The method of claim 51 wherein the mutation in the second polypeptide is a serine residue at E333.
54. The method of claim 51 wherein the second polypeptide comprises two of said mutations.
55. A chimeric protein comprising a first and a second polypeptide wherein the first polypeptide is a Factor VIIa polypeptide and the second polypeptide is an Fc region of a human immunoglobulin IgG1, wherein the Factor VIIa polypeptide contains at least one mutant residue which reduces blood coagulation activity relative to wild-type Factor VIIa.
56. The chimeric protein of claim 55 which is in the form of a dimer.